



A Rare Case of Lymphatic Filariasis

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Report

ABSTRACT

Lymphatic filariasis is a parasitic infection caused by *Wuchereria bancrofti*, *Brugia malayi* and *Brugia timori*. Asymptomatic microfilaria, acute lymphatic filariasis, chronic lymphatic filariasis, tropical pulmonary eosinophilia are the different presentations of lymphatic filariasis. Systemic manifestation can involve joint, kidney, heart and nerve. This article is a case report of lymphatic filariasis with a rare presentation of anasarca and nephritic syndrome.

Keywords: Filariasis; nephritic syndrome; Anasarca.

1. INTRODUCTION

Filariasis is one of the oldest and most neglected tropical diseases. About 1.4 billion people worldwide are threatened with filariasis, more than 120 million people are currently infected. After leprosy, filariasis is the second most common cause of long-term disability [1]. India, China and Indonesia together bear the burden of two-thirds of all caseloads [2]. In India, filariasis

is prevalent in 17 provinces and in 6 unions that affect some 31 million people [3]. It is most common in Orissa, where its prevalence is highest in the coastal region of Cuttack. Although it is a common disease, there are no detailed studies showing that the kidneys may be involved in filariasis. However, there are separate reports of kidney involvement [4]. Historical changes of kidney involvement such as mesangioproliferative glomerulonephritis, acute

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eosinophilic glomerulonephritis and membranous glomerulopathy have been documented [4]. Structural glomerular changes and the incorporation of immune complexes of immunoglobulin G, immunoglobulin M and C3 in mesangium, capillary wall or glomerular membrane have been reported.

2. CASE REPORT

A 35-years-old male presented with complaints of anasarca for 8 months and difficulty in breathing aggravated on exertion. Patient also complained of cough with expectoration. Patient also complained of bilateral lower limb swelling which was gradual in onset following which he developed facial puffiness followed by abdominal swelling. History of decreased urine output for 1 month. No history of paroxysmal nocturnal dyspnoea, palpitations, jaundice, hematemesis and melaena. Past history of treatment with DEC for pedal edema before 8 months. On examination, facial puffiness with swollen eyelids, pallor and bilateral pitting pedal edema extended up to both knees. Vitals were within normal range. On abdominal examination, patient had massive ascites, scrotal and penile swelling. On auscultation, patient had bilateral expiratory wheeze and coarse crepitation. Central nervous system and cardiovascular system had no abnormality.

Investigations showed reduced haemoglobin (9 mg/dl), eosinophilia (E-34), peripheral smear showed normocytic and normochromic anemia, coagulation profile was normal. Liver function test showed normal bilirubin and enzyme level, total protein was 3g/dl and albumin were 1.3g/dl. Renal function test showed values within normal range. Urine routine had high proteinuria, 3+ albumin, 20-24 RBC and 3-4 puss cells and 24 hours urinary protein was 4560mg/day. Stool examination showed no parasite cysts/ova. Serology (HIV, HBsAg and Anti-HCV) were non-reactive. ASO titer was normal. Ultrasound abdomen showed ascites and hepatomegaly. Ascitic fluid analysis showed albumin level of 0.4g/dl and serum ascites albumin gradient were 0.9. Patient was started on ACE inhibitors and diuretics. In view of eosinophilia and past history of treatment with DEC, peripheral smear done for filarial parasite, showed no parasite and rapid filarial antigen test was positive. Kidney biopsy showed subepithelial deposits of IgG and complement features suggestive of membranous glomerulonephritis.

Patient was treated with diethyl carbamazepine (DEC) daily thrice for 21 days and antibiotics for 14 days. Following treatment, eosinophilia reduced and eosinophil count normalized, urine routine showed 1+ proteinuria, serum protein increased to 1.6g/dl, 24 hours urine protein started reducing to 300mg and 68 mg after 15 days and 30 days respectively. Pedal edema, facial puffiness, scrotal and penile swelling reduced and 5 kg weight reduction occurred after treatment for 1 month. Patient was discharged and followed up regularly.

3. DISCUSSION

Association of glomerular disease and filariasis has been studied many times. Immune complex mediated glomerulonephritis and filariasis has been reported. Presence of granular deposits of IgG and C3 in immunofluorescence microscopy and decreased complement factors indicate glomerulonephritis due to immune complex deposition. Different histopathological abnormalities of glomerulonephritis can be present in filariasis. Membranous glomerulonephritis, membranoproliferative and mesangial glomerulonephritis are other types of glomerulonephritis seen in filariasis. Presence of anasarca and glomerulonephritis is a very rare presentation in filariasis. It is important to know about the extra lymphatic manifestations of Bancroftian filariasis for proper diagnosis and treatment.

4. CONCLUSION

Lymphatic filariasis is a spectrum of illness and can manifest as, asymptomatic microfilaria, acute lymphatic filariasis, chronic lymphatic filariasis, tropical pulmonary eosinophilia and some systemic manifestations which involves joint, heart, kidney, nervous system etc. From the above reference nephritic syndrome and anasarca should be considered for the differential diagnosis for lymphatic filariasis to prevent mortality and morbidity. The association between filariasis and glomerular disease is discussed by recent studies [5-8]. However its difficult to diagnosis intially because of association with co-infection of malaria and hepatitis. Minimal change disease, focal and segmental glomerular sclerosis is rare presentation of filariais.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our

area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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